

# Exposure to Polycyclic Aromatic Hydrocarbons among Dutch Children

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We determined the urinary 1-hydroxypyrene (1-HP) concentration and the creatinine-adjusted 1-HP concentration in 644 randomly selected Dutch children, aged 1–6 years and living in five areas with roughly different levels of polycyclic aromatic hydrocarbons (PAHs) in soil and ambient air. The presence of other factors that might influence the exposure to PAHs was studied using a questionnaire. To evaluate the reliability of a single urinary 1-HP determination, measurements were repeated after 3 weeks for approximately 200 children. The mean urinary 1-HP content of the total study population was 2.06 nmol/l. This varied from 1.58 nmol/l in the reference area (Flevoland) to 2.71 nmol/l in the valley of the Geul. Only indoor sources of PAHs showed a small, positive association with urinary 1-HP. The urinary 1-HP concentrations of children from the valley of the Geul were higher ( $p < 0.01$ ) and those of children from a suburb of Amsterdam were lower ( $p < 0.01$ ) than those of children from the reference area. The possible ambient environment-related differences were probably too small to be detected in the variations of the intake of PAHs from the daily diet. The reliability of a single 1-HP measurement was low. Similar results were obtained with the creatinine-adjusted data. In one neighborhood built on coal-mine tailings, the urinary 1-HP content in children was weakly but positively associated with the PAH content in the upper soil layer of the garden of their homes. However, this association was not found for the children from the other neighborhood built on coal-mine tailings and with similar PAH levels in soil. **Key words:** biological monitoring, children, 1-hydroxypyrene, polycyclic aromatic hydrocarbons, soil contamination. *Environ Health Perspect* 104:530–534 (1996)

Polycyclic aromatic hydrocarbons (PAHs) are compounds with two or more benzene rings that have long polluted the ambient environment and food. The dispersion of PAHs into the ambient environment is the result of natural and anthropogenic processes. In the Netherlands, anthropogenic sources such as the combustion of fossil fuels and waste incineration are important. The local levels of PAHs in ambient air are determined by emissions from traffic, open hearths, oil-fired heaters, and sources in other countries (1–5).

The present exploratory study examines the exposure to PAHs by biological monitoring of young children living in areas with roughly different levels of PAHs in the ambient environment. The differences in PAH levels in soil and air were based on studies of the environmental levels of PAH in the Netherlands carried out previously (1,2,4,5). A biomarker developed by Jongeneelen et al. (6), the 1-hydroxypyrene (1-HP) concentration in urine, was used. We were interested in personal exposure to PAHs as estimated with the urinary 1-HP levels in children and particularly in the influence of soil contamination with PAHs on these levels. In a considerable number of studies, the utility of 1-HP, a metabolite of pyrene, as a biomarker of occupational

exposure to PAHs has been described (7,8). The exposure to PAHs can be estimated with this biomarker if the proportion of the pyrene content in the mixture of PAH is constant (7). The method was also sufficiently sensitive to detect differences in low-level exposures to PAHs by windsurfers surfing on PAH-contaminated surface water (9). As the method had not yet been used with young children, a subgroup of the study population was sampled twice to obtain information on the reliability of a urinary 1-HP measurement. Since the execution of this study, other results of urinary 1-HP measurements in children have become available and will be considered in the discussion.

## Materials and Methods

The 1-HP concentration in urine was determined in groups of randomly selected, young, white children, ages 1–6 years, living in five areas with different levels of PAHs in soil and ambient air. The presence of other factors that might influence the exposure to PAH was evaluated using a questionnaire (66 items). Approximately 200 children, evenly distributed over the study areas, were sampled twice with an interval of 3 weeks. At this time, a shortened version of the questionnaire was completed by the parents.

Environmental levels of PAHs in soil and ambient air in the Netherlands has been studied previously (1,2,4,5). Inner-city, suburban, and rural areas were included in the present study, as well as two residential areas built on soil contaminated with PAH (10–13). The study areas were 1) city center: districts in the inner cities of Amsterdam and Rotterdam, which have soils historically contaminated with PAHs and elevated levels of PAHs in ambient air due to the high density of traffic; 2) suburb: one suburban district in each of these two cities, all built on unpolluted soil with lower traffic densities than in the city centers; 3) soil contamination: two neighborhoods (Laura, Eikske) built on the tailings of former coal mines contaminated with PAHs, and levels of PAHs in ambient air possibly influenced by source areas in Germany and Belgium; 4) control: a number of villages in the valley of the Geul (E.S.-Limburg) as control area for 3; and 5) rural: Flevoland, a thinly populated, agricultural polder province, as an unpolluted reference area.

The questionnaire, completed by the parents, obtained information on the recent consumption of food articles with a relatively high PAH content, the presence of indoor and outdoor sources of PAHs, hand-to-mouth behavior and play habits of the child, and the education level and profession of the parents.

Children age 1–6 years were selected randomly from the population registries of the municipalities concerned. In Amsterdam the registries of the Youth Health Department were used; the completeness of these files, which are continuously updated, is higher than 95%. All children had to reside at least 2 months at their present address. We aimed to study roughly 700 children, about 200 living in the old city centers, about 200 in the reference area

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(Flevoland), and about 150 living in E.S.-Limburg and the suburbs each. The size of the population sample was determined by an assumed response of 40% in Amsterdam and Rotterdam and a response of 50% in the other study areas. The parents of the children were approached by letter from their respective health services.

All children participating were sampled June–July 1992 before the start of school vacation. All repeat samples were also obtained before school vacation. Parents of participating children signed an informed consent. During the entire sampling period, weather conditions were conducive for outdoor play (e.g., high temperatures and no precipitation). Urine samples were collected at home by the parents in dedicated containers, usually in the morning and in the early afternoon. The adjustment of the urinary 1-HP concentration with the creatinine concentration was not systematically biased by the time of urine collection, as time of collection did not contribute to the explained variance in the multiple regression. For children not yet toilet-trained, a special urine bag (U-Bag, Hollister) was used. The urine samples were refrigerated within 3 hr and frozen at -20°C within 12 hr.

Urine samples were analyzed in the Laboratory of the Department of Toxicology at the University of Nijmegen. 1-Hydroxypyrene in urine was determined by HPLC with fluid gradient and fluorescence detection and related to the creatinine level. The analytical method for determination of 1-hydroxypyrene has been described elsewhere (6). The detection limit of the method is 0.09 nmol 1-HP/l urine. Urinary creatinine concentrations were determined by measuring photometrically at 500 nm the concentration of the colored complex that results from the reaction of creatinine with alkaline picrate. The detection limit of this method is 0.2 mmol creatinine/l urine.

The precision of the analysis of urinary 1-HP was 13.1% (intraday variation,  $n = 56$  duplicate analyses) and 19.6% (interday variation,  $n = 53$  duplicate analyses); we assessed the accuracy by analyzing one or two composite samples with a 1-HP concentration of 35 nmol/l during each series of analyses. The accuracy was 5.6% ( $n = 72$  analyses). For urinary creatinine, the precision was better than 5%. We assessed the accuracy by analyzing one or two composite urine samples with a creatinine concentration of 15 mmol/l during each series of analyses. The variation was 5.1% ( $n = 72$  analyses), and the between-laboratory variation was 12.4% ( $n = 10$  samples).

To facilitate comparisons with urinary 1-HP concentrations published in the litera-

ture, we present our data both adjusted and not adjusted for urinary creatinine content.

We used SPSS PC Plus, version 4.01 (SPSS Inc., Chicago), for descriptive statistics, mostly frequency tables. Multiple linear regressions were carried out with the natural log-transformed 1-HP concentrations or the creatinine corrected 1-HP concentrations as dependent variables. The study areas were incorporated in the model as dummy variables with Flevoland as reference. With the results of the repeat measurements, the reliability coefficients were calculated according to Armstrong et al. (14). In this method, the term “reliability” refers to the reproducibility of a measure; that is, how consistently a measurement can be repeated on the same subjects. The closer the value of the reliability coefficient is to one, the higher the predictive value of the measurement.

## Results

Cooperation with the study was offered by 50.3% of the parents in reply to the 1821 letters sent. This varied from 46.3% in the suburb of Amsterdam to 63.3% in the two neighborhoods built on coal-mine tailings. In all study areas the response was higher than expected and, consequently, not all parents were invited for an appointment. Completed questionnaires were received for 716 children, and from 667 children a urine sample was obtained. Out of this group, 644 children had a creatinine concentration in urine above the detection limit (0.2 mmol/l). The mean age of the children by study area varied from 3.2 to 3.7 years. Repeat samples, with an interval of 3 weeks, were obtained from 203 children evenly distributed over the study areas. For 199 children, the creatinine content in both samples was higher than the detection limit.

Within the suburb study area the mean 1-HP concentration of children from the suburb of Amsterdam was lower ( $p < 0.01$ ) than that of the children from the suburb of Rotterdam. Within the other study

areas, no statistically significant differences of the urinary 1-HP concentrations between subgroups of children were found. Therefore, the results are presented by six study areas.

After natural log transformation, the 1-HP concentrations were normally distributed. Table 1 shows the geometric mean concentrations of 1-HP in urine and the geometric means of the creatinine-corrected 1-HP concentration by age and sex. The first measurement included the total study population, while the repeat measurement was carried out in a part of the study population.

Geometric mean urinary 1-HP concentrations ranged from 0.38 nmol/l to 1.43 nmol/l in the different age classes. The 1-HP concentrations correlated positively with age in the total study group and in the repeat group (Spearman  $r^2$  0.26,  $p < 0.001$  and 0.24,  $p < 0.001$ ). One year olds had lower ( $p < 0.05$ ) urinary 1-HP concentrations than the 2 year olds, who had lower 1-HP concentrations than the other age groups, between which the 1-HP concentrations did not differ. Similar results were obtained for the group with the repeat measurement. The creatinine-corrected 1-HP concentrations in urine did not differ between age groups. The 1-HP concentrations did not differ between boys and girls, except the slightly higher ( $p < 0.05$ ) creatinine-corrected 1-HP concentrations of girls in the total study population. Table 2 shows the urinary 1-HP concentrations and the creatinine corrected 1-HP concentrations by study area.

The mean urinary 1-HP content in the total study population was 2.06 nmol/l ( $n = 644$ ). The 1-HP content varied from 1.58 nmol/l in the reference area in Flevoland to 2.71 nmol/l in the valley of the Geul. The geometric mean urinary 1-HP concentrations varied from 0.64 nmol/l (Amsterdam suburb) to 1.73 nmol/l (valley of Geul). The maximal 1-HP concentrations varied from 9.85 nmol/l in the reference area to 47.3 nmol/l in the two neighborhoods built on coal-mine tailings. The maximal 1-HP

**Table 1.** Geometric means of urinary (creatinine-corrected) 1-hydroxypyrene (1-HP) concentrations by age and sex in the total study group and in the group with a repeat measurement

Sample	Age (years)						Sex		<i>n</i>
	1	2	3	4	5	6	Male	Female	
Total study group									
1-HP (nmol/l)	0.38	0.75	1.33	1.14	1.36	1.43	0.90	1.06	667
1-HP/CR (μmol/mol)	0.19	0.22	0.25	0.20	0.20	0.18	0.19	0.22	644
Repeat group									
1-HP (nmol/l) 1st measurement	0.31	0.73	1.59	1.25	1.45	2.13	1.21	1.28	203
2nd measurement	0.29	0.59	0.90	1.13	1.09	1.43	0.81	1.07	203
1-HP/CR 1st measurement	0.10	0.17	0.26	0.20	0.21	0.27	0.20	0.22	199
2nd measurement	0.10	0.15	0.17	0.17	0.14	0.17	0.14	0.17	199

CR, creatinine.

value in the group of children from the suburb of Amsterdam was 42.6 nmol/l. Similar results were obtained for the creatinine-corrected urinary 1-HP values.

The multiple linear regression model incorporated the results of the questionnaire on factors that might influence exposure to PAHs as six aggregated explanatory variables: 1) specific sources in the daily diet, 2) indoor sources, 3) outdoor sources, 4) hand-to-mouth behavior, 5) outdoor play, and 6) the level of education and profession of the parents. The study areas were included as dummy variables in the model, with the children from Flevoland as the reference population. Only approximately 3% of the variance of the 1-HP concentrations was explained by the variables in the questionnaire. Indoor sources of PAHs [e.g., open hearths, multi-burners (stoves intended to burn wood, but often used to burn many kinds of combustible materials), smoking habits] showed a small positive association with the urinary 1-HP content (regression coefficient = 0.13,  $p < 0.05$ ). The explained variance of the model was 11% with the creatinine-corrected 1-HP concentrations as dependent variable.

The urinary 1-HP concentrations of the children from villages in the valley of the Geul were higher ( $p < 0.01$ ) and those of the children from the Amsterdam suburb were lower ( $p < 0.01$ ) than those of the children from the reference area, Flevoland. The urinary 1-HP concentrations of the groups of children from the other study areas did not differ from those of the reference group. In the group with the repeat

measurement, the 1-HP concentrations of children from the reference area did not differ from those of the children from the other study areas.

The excretion mechanism of 1-HP might be altered when the urine sample is very concentrated or very diluted (15). The limits for a reliable correction for creatinine for children are unknown. We repeated the statistical analysis, excluding urine samples with a creatinine content lower than the 5th percentile and higher than the 95th percentile (calculated over the total study population). The results did not differ from those obtained with the analyses without this restriction.

A repeat measurement with an interval of 3 weeks was obtained for approximately 200 children. After 3 weeks the results are independent because the biological half-life of 1-HP is roughly 24 hr (8). The correlation between the 1-HP concentrations of the children was 0.29 ( $p < 0.001$ ) and 0.40 ( $p < 0.001$ ) for the creatinine-corrected concentrations. The predictive value of a urinary 1-HP measurement of a child was evaluated by calculating the reliability coefficient according to Armstrong (14). The closer the value of the reliability coefficient is to one, the higher the predictive value of the measurement. Both the uncorrected and the corrected 1-HP concentrations were used. As shown in Table 3, the reliability coefficients were low for the total group and varied from 0.03 in the reference group to 0.54 in the suburb of Amsterdam.

In two neighborhoods (Laura and Eikske) in E.S.-Limburg, built on the tail-

ings of former coal mines, the presence of PAHs in soil was investigated by chemical analysis (10–13). The PAH concentrations were known for representative soil samples taken from the upper soil layer (first 50 cm) in the front and back gardens of the homes. In Laura, this information was available for 33 children and in Eikske for 37 children participating in our study. The PAH concentration in soil was entered as an explanatory variable in the multiple linear regression model described above. The PAH concentrations in soil were expressed as 1) the pyrene content and 2) the sum concentration of the PAHs on the list ( $n = 16$ ) of the U.S. Environmental Protection Agency (2). This list includes the concentrations of naphthalene, acenaphthene, acenaphthylene, fluorene, phenanthrene, anthracene, fluoranthene, chrysene, benzo[*a*]anthracene, pyrene, benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, dibenzo[*ah*]anthracene, indeno[1,2,3-*cd*]pyrene and benzo[*ghi*]perylene.

In Eikske and Laura, strong correlations ( $r > 0.89$  and  $r > 0.95$ ) were observed between the concentrations of pyrene and the sum concentrations of EPA PAHs in the soil samples. In Eikske the proportion pyrene was on average approximately 12% (SD ~5%) of the sum EPA PAHs in the soil samples of the back and front gardens, while in Laura the proportion of pyrene amounted on average to approximately 20% (SD ~5%) of the sum EPA PAHs. The results of multiple regression analyses with the urinary 1-HP concentration as dependent variable are presented in Table 4.

**Table 2.** Urinary (creatinine-corrected) 1-hydroxypyrene (1-HP) concentrations by study area

Study area	1-HP (nmol/l)					1-HP/creatinine (μmol/mol)					<i>n</i>
	AM	SD	Min	Max	GM	AM	SD	Min	Max	GM	
Amsterdam/Rotterdam center	2.05	2.51	0.05	16.22	0.99	0.34	0.38	0.00	2.50	0.20	198
Amsterdam suburb	1.84	5.25	0.05	42.59	0.64	0.32	0.89	0.01	7.15	0.11	66
Rotterdam suburb	2.69	3.47	0.05	17.38	1.45	0.35	0.37	0.00	1.94	0.23	62
Eikske/Laura	2.29	5.52	0.05	47.26	0.99	0.37	0.77	0.03	6.39	0.21	77
Valley of Geul	2.71	2.78	0.05	14.92	1.73	0.43	0.40	0.03	1.58	0.30	66
Flevoland	1.58	1.47	0.05	9.85	1.06	0.30	0.24	0.02	1.57	0.23	175
Total	2.06	3.31	0.05	47.26	1.06	0.34	0.49	0.00	7.15	0.21	644

Abbreviations: AM, arithmetic mean; GM, geometric mean.

**Table 3.** Reliability coefficients (14) by study area

Study area	Urinary 1-HP ( <i>n</i> )	1-HP/creatinine ( <i>n</i> )
Amsterdam/Rotterdam center	0.28 (73)	0.36 (69)
Amsterdam suburb	0.54 (15)	0.26 (15)
Rotterdam suburb	0.38 (16)	0.43 (16)
Eikske/Laura	0.45 (28)	0.53 (28)
Valley of Geul	0.43 (25)	0.45 (25)
Flevoland	0.03 (46)	0.34 (45)
Total	0.28 (203)	0.37 (198)

1-HP, 1-hydroxypyrene.

**Table 4.** Results of multiple linear regression with the soil concentration of PAHs in home gardens of children in Eikske and Laura as one of the explanatory variable and urinary 1-hydroxypyrene as the dependent variable

	Eikske ( <i>n</i> = 37)			Laura ( <i>n</i> = 33)		
	β	SE	<i>p</i>	β	SE	<i>p</i>
Pyrene						
Back garden	0.16	0.11	0.07	0.11	0.17	0.28
Average of back and front garden	0.12	0.14	0.19	0.08	0.24	0.38
Sum EPA PAH <sup>a</sup>						
Back garden	0.30	0.12	0.09	-0.10	0.15	0.27
Average of back and front garden	0.23	0.14	0.055	-0.06	0.21	0.39

<sup>a</sup>Sum of PAHs listed by EPA (2).

In Eikse the regression coefficients were small, but all were positive and most reached a (marginal) statistical significance. In Laura only small and mostly negative (statistically nonsignificant) regression coefficients were obtained. Multiple linear regression with the creatinine-corrected 1-HP concentrations gave similar results.

## Discussion and Conclusions

Exposure to PAHs can be estimated with the urinary 1-HP concentration if the proportion of pyrene in the mixture of PAHs is constant. Pyrene forms a relatively large proportion of the PAHs in food and ambient air in the Netherlands (1,2,16,17). Important industrial point sources in the Netherlands are the production of coke and aluminum (carbon electrodes) (1,2). These locations were avoided in this study. The important diffuse sources are traffic and the incineration of coal and oil. Local sources of PAHs in ambient air are oil heaters, multi-burners, and traffic. A large proportion of the local level of PAHs is determined by source areas in other countries (1,2). The daily diet contributes substantially to nonoccupational exposures (16,17–19). Within the Netherlands, the results of measurements of foods do not indicate systematic regional differences in the proportion of pyrene in the PAH profile of the daily diet (16,20). Large-scale production and distribution of food products prevail in the Netherlands. As expected, the average intake of PAHs by food products, as assessed with the questionnaire, did not differ between study areas. As far as we know, the PAH profiles in ambient air, locally dependent on traffic density and oil and coal heating, also do not show systematic regional differences within the Netherlands (1,2,21). The study design accounted for the possible influence of industrial source areas in Belgium and Germany on the PAH content of the ambient air in E.S.-Limburg. Therefore, we assumed that a distinct influence of environmental contamination with PAHs on the exposure to PAHs could be detected by comparing urinary 1-HP concentrations.

In the total study population, the geometric mean creatinine-corrected 1-HP concentration of girls was somewhat higher ( $p < 0.05$ ) than that of boys. A study of children in Poland showed the opposite (22). In our study, the urinary 1-HP concentrations did not differ between boys and girls. The geometric mean urinary 1-HP concentrations increased ( $p < 0.001$ ) with age in the total study group ( $r = 0.26$ ) as well as in the group with a repeat measurement ( $r = 0.24$ ). However, the correlation was low. The creatinine-corrected 1-HP concentra-

tions did not show a trend with age. The excretion of creatinine depends on muscle mass and thus age. The mean urinary creatinine concentrations did not differ between girls and boys, but did correlate with age ( $r = 0.4$ ,  $p < 0.001$ ); therefore, the relation of the creatinine-corrected urinary 1-HP concentration with age disappears. We conclude that exposure to PAHs of the children 1 and 2 years of age was lower than that of older children in the study group and that the exposure to PAH tends to increase with age.

Only indoor sources of PAHs (smoking habits of household members, multi-burners, profession) were associated with the urinary 1-HP concentration. The other explanatory variables like outdoor sources of PAHs or the intensity and location of outdoor play did not influence the 1-HP concentration. The significance of these findings is limited because exposure to PAHs assessed with the questionnaire was graded based on published results of measurements (1,2,20) and not on actual measurements of contact media.

The urinary 1-HP concentrations of the children from the valley of the Geul were higher and those of children from the Amsterdam suburb were lower than those of the children from the reference area, Flevoland. This result did not conform to our expectation that the urinary 1-HP content would increase from Flevoland through the suburbs of Amsterdam and Rotterdam to the districts in the old inner-cities of Amsterdam and Rotterdam. Similar results were obtained when the 1 and 2 year olds were excluded from the statistical analyses. The daily diet is the dominant source of exposure to PAHs (17–19). The possible ambient environment-related differences in exposure to PAH were probably too small to be detected in the variations in the intake of PAHs by the daily diet.

As shown in Table 3, the reliability of a single measurement of a child was low. This indicates that the intake of PAHs varies quickly over time and that comparisons of single urinary 1-HP values are not informative. However, with comparisons of the 1-HP concentrations at the group level, one might expect these variations in time to cancel each other, allowing cross-sectional comparisons of the exposure to PAHs of groups.

In this study the soil contamination with PAHs of residential gardens resulted in a positive association with the urinary 1-HP concentration of children in one neighborhood (Eikse), while for the children from the other neighborhood no such association was detected. The different outcome for the two neighborhoods is not explained by dif-

ferences of the PAH concentrations in the gardens of the homes. The highest concentrations of PAHs were found in the back gardens, where the pyrene concentrations ranged up to 24 mg/kg dry weight (mean 3.4 mg/kg) in Laura and up to 17 mg/kg dry weight (mean 1.5 mg/kg) in Eikse. The mean sum EPA PAH concentrations in the back gardens were 27 mg/kg dry weight (maximum 189 mg/kg) in Laura and 12 mg/kg dry weight (maximum 96 mg/kg) in Eikse (10–13). A possible explanation of the association between PAH content in soil and urinary 1-HP found in Eikse is that in Laura more measures to restrict direct contact of children with soil have been taken than in Eikse. This fits the finding that multiple regression with the results of both neighborhoods showed an influence ( $p < 0.01$ ) of neighborhood. However, these results might also be explained by chance alone.

Some published reports have become available of other studies of the urinary 1-HP concentrations in children. Eight- and 9-year-old children in Bytom, Upper Silesia, an industrial area in Poland heavily polluted with PAHs, had higher urinary 1-HP concentrations than our study groups. Girls ( $n = 76$ ) had a mean urinary 1-HP concentration of 6.3 nmol/l (SD 4.9) and the mean 1-HP concentration of boys ( $n = 72$ ) was 7.6 nmol/l (SD 9.3) (22). The mean urinary 1-HP concentrations up to 1.0 nmol/l of Japanese children (10–12 years) attending elementary schools in urban areas with and without a high traffic density suggest a somewhat lower exposure to PAHs than the children in our study (23). In Beijing, which has elevated PAH levels in ambient air, a small group ( $n = 15$ ) of schoolchildren had a creatinine-corrected mean 1-HP concentration of 0.52  $\mu\text{mol/mol}$  (24). These studies illustrate that ambient environment-related differences in the exposure to PAHs can be detected by measuring urinary 1-HP content.

An established health limit value for the urinary 1-HP concentration is lacking. Recently, Buchet et al. (25) studied the feasibility of an extensive panel of biomarkers to detect early effects of PAH exposure of workers in coke ovens and a graphite electrode plant. They suggested that to avoid early health effects, urinary 1-HP concentrations should be kept below 1.4  $\mu\text{mol}$  1-HP/mol creatinine in these workers, mainly exposed by the pulmonary and dermal routes. In our study, 15 (2.3%) children had a single urinary 1-HP concentration higher than 1.4  $\mu\text{mol/mol}$ , while in the group sampled twice, 3 (1.5%) children had an average urinary 1-HP concentration higher than 1.4  $\mu\text{mol/mol}$ , and only 1

child had a urinary 1-HP level higher than 1.4  $\mu\text{mol/mol}$  at both measurements. But, as these children are exposed predominantly by the oral route, the observations on workers might not be applicable (Buchet JP, personal communication).

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## EPA NHEERL TO HOST OPEN HOUSE

You are invited to attend an Open House sponsored by EPA's National Health and Environmental Effects Research Laboratory (NHEERL) on June 5, 1996. It will be held at the EPA Environmental Research Center at Alexander Drive and Highway 54, Research Triangle Park, North Carolina. Posters explaining research conducted by NHEERL investigators will be available for viewing from 9:00 a.m. to 3:30 p.m. Brief presentations describing NHEERL's research program will also be given. Lunch will be available for a small fee. Registration begins in April. For more information call (919) 541-5193.